*Proceedings of the 2012 Winter Simulation Conference*
*C. Laroque, J. Himmelspach, R. Pasupathy, O. Rose, and A. M. Uhrmacher, eds.*

# Modelling the Minsky triad: A framework to perform reflexive M&S studies

Bruno Bonté
Lab. of Complex Systems Eng. (LISC)
IRSTEA - 24, av. des Landais BP 50085
63 172 Aubière Cedex, France

Jean-Pierre Müller
Renewable resources and Env. Man. (GREEN)
CIRAD - Campus de Baillarguet
34398 - Montpellier France

Raphaël Duboz
Animals and Integrated Risk Management (CIRAD-AGIRs)
Computer Science and I-Man. (AIT-CSIM)
AIT, 12120 Pathumthani Thaïlande

## ABSTRACT

In this paper, we propose a general framework to evaluate models of systems that are ill defined, incompletely known, and furthermore, which cannot be experimented in real conditions, such as the economical systems at the country scale, epidemics (for obvious ethical reasons) or any natural disasters, for instance where human lives are the main issue. Our framework relies on the generic Marvin Minsky's definition of a model and its specification in the frame of the Theory of Modelling and Simulation, initiated by B.P. Zeigler. Such a dynamic system implementation of the Marvin Minsky's model definition, we called the Minsky triad model, enables to address original questions. The Minsky triad model is a coupled model composed of the model of a real system, the model of this later model, and, in between, the model of the user of the later model. We argue that the Minsky triad model is very promising as a framework to design and to evaluate decision support systems for crisis management.

## 1 INTRODUCTION

An important preliminary activity in modelling and simulation is to gather data from the target system we are interested in. Such data are used to build, to calibrate and to validate the model. Thereafter, we can learn from the model or forecast behaviours in order to be able to make decisions and to take actions on the system under study. A problem arises when the target system cannot be experimented for any reason. For instance, if we consider an epidemic in a human or animal population, we cannot experiment such a system for obvious moral and ethical reasons. Similar problems arise in many situations where human lives are the main issue. More generally and less dramatically, we can say that the systems defined at the ecological, economical or social scales (Socio-Eco-Systems, SES) cannot be experimented in most cases.

A solution is to model *a priori* such systems, using a non validated model (or validated with previous similar situations) to support the decisions when a new situation occurs. In that case, we do not know if the model we are using would provide useful answers regarding this new situation. In this paper, we propose a framework to address such an issue. The problem is to evaluate if a given a model enables to make the appropriate decision in situations that have not been previously encountered. Furthermore, to be complete, such a framework should consider the interdependency between the target system dynamics and the actions the decision maker decides upon based on his model. Indeed, in the context of SES systems, the objective of the decisions is to control the future trajectories of the system by acting on it, modifying in return the future trends of the system dynamics.

The framework we propose in this paper derives from the Marvin Minsky's definition of what is a model. In 1965, M. Minsky said: "To an observer B, an object A* is a model of an object A to the extent that B can use A* to answer questions that interest him about A" (Minsky 1965). Starting from that

definition, we call the "Minsky triad" T, the three entities A, B and A*. Figure 1 page 2 represents this triad and the relations between the three entities that compose it. We call $\rho_o$ and $\rho_m$ the relations between the observer and the object and between the observer and the model respectively. Such a representation is the first step towards a systemic conception of the triad.

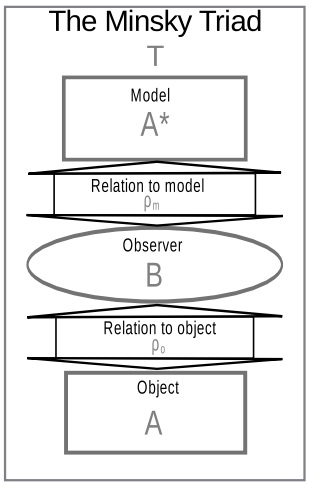


Figure 1: The Minsky triad $T$.

The next section will present the general conceptual framework. Then we will present more specifically the model of the triad using the Theory of Modelling and Simulation. We will illustrate the use of this framework on a specific case. We will then discuss the obtained results before to conclude.

## 2 THE GENERAL FRAMEWORK

The figure 1 illustrates the classical relation among a model, the target system and the user of the model, as a Minsky triad $T$. The main idea of this work is to model the Minsky triad itself, leading to a reflexive representation to address questions related to the use of models. Therefore, in order to study the interactions among the three entities of the triad, we propose to build a model $T^*$ of the triad $T$. Doing that, we create a new triad in which the target system is the triad $T$, the observer is the researcher $C$ and the model is the model $T^*$. This last triad is the general conceptual framework that we propose for model evaluation. We note $T'$ this framework and present it in figure 2. In order to address questions about the use of model $A^*_B$, $C$ build a model $T^*$ of the triad $T$. $\rho_{o\_c}$ is the question $C$ has on $T$. $\rho_{m\_c}$ is the experimentation (i.e. the simulations) $C$ performs on $T^*$. In this framework, the question of $C$ will be not be about the model $A^*_B$, but about the *use* of the model $A^*_B$ by $B$ as far as the model $A^*_C$ sufficiently represents the behaviour of $A$. Consequently, the $T^*$ model must contain a model of each entity and relation present in the triad $T$: $A_C$* is a model of entity A for observer $C$ and $A^*_B$ is a model of entity $A$ for observer $B$, $A^{**}$ is both a model of $A$ for $B$* and a model of $A_B$* for the observer $C$. In general, the model $A^{**}$ is identical to the model $A*_B$. Finally, $\rho^*_o$ is the model of teh relation $\rho_o$ between the observer $B$ and the target system $A^*$ and $\rho^*_m$ is the model of teh relation $\rho_m$.

Having this general framework, we can use the concepts and formalisms from the Theory of Modelling and Simulation (TMS) initiated by B.P. Zeigler (Zeigler, Kim, and Praehofer 2000) to specify the model of the Minsky triad $T^*$, and to design and implement the corresponding simulator. Indeed, we could directly build a morphism between the entities of the Minsky triad including their relations on the one hand, and the models specified using the Discrete EVent systems Specification (DEVS) formalism on the other hand. In our framework, the model of the observer $B^*$ has to manipulate the model $A^{**}$ in order to take decisions on the model of the target system $A^*_C$. The main difficulty here is in the formalisation of the use of a model by

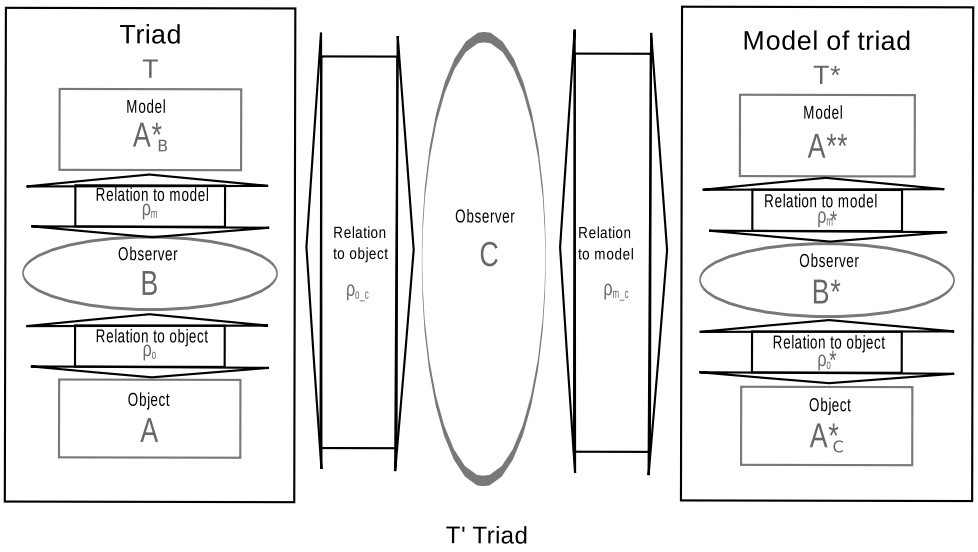


Figure 2: The general framework $T'$.

another model. Indeed, when an observer uses a model to make decisions, the model has its own simulation time line, which is different from the time line of the model user. To tackle this particular problem, we have proposed the use of the recursive simulation technique described in a previous study (Bonté, Duboz, Quesnel, and Müller 2009). This technique is combined to the observer $B^*$ dynamics using the concept of experimental frames. In the TMS, any question about a dynamic system can be described using an experimental frame (Zeigler, Kim, and Praehofer 2000, Traore K. Mamadou 2006). The experimental frame specifies the system environment, or "context of interest". It basically specifies the input signals sent to the system (or to the simulator) and the observation policy i.e. the simulation outputs that are monitored. For example, any validation process consists in comparing the behaviour of the system with the behaviour of the model within the experimental frame, using the simulation outputs.

In the following, we present a possible specification of the $T^*$ model using TMS and more specifically DEVS.

## 3 THE T* DEVS MODEL

The DEVS formalism allows to specify the $T^*$ model as a generic hierarchical structure. Within this structure, some models of sub-processes are generic and others can be specified and reused at will thanks to the modularity feature of the DEVS formalism.

The $A_C$* model is a DEVS model. The $\rho_o$ relation between the observer and the target system is formalised as a Sub-Process for Observation and Control (SPOC). Its model is $\rho_o$*. For instance, it can be composed of a model of observation and a model of action coupled together.

Considering $A_B$* is a model of a dynamic system, we can design a simulator to simulate it. We consider that a simulation is a virtual experiment. Doing so, the $\rho_m$ relation between the observer and the model is considered as an experimental process performed on the $A_B$* model by $B$. The corresponding $\rho_m$* model in figure 2 is consequently called the Experimentation Process Model (EPM). It models the $\rho_m$ relation between the observer and the model. However, we must distinguish between a simulation model and a dynamic system. B.P. Zeigler gives the following definition of a dynamic system simulation model: "A simulation model [...] is a set of instructions, rules, equations or constraints allowing to generate an Input/Output (IO) behaviour". In our case, one can imagine the $A_B$* model as a set of rules or equations. Thus, the dynamic system model describes a dynamic system behaviour, but it is not a dynamic system

itself until we simulate it. By opposition, a dynamic system actually *generates* IO behaviour. Therefore $A_B$* cannot directly interact with any triad sub-systems. These equations or rules cannot be coupled with the other DEVS sub-systems. Likewise, the EPM cannot directly interact with the $A^{**}$ model by sending events to it because $A^{**}$ stays a model (a set of instructions, rules etc., and not a physical dynamic system) within the $T^*$ model. To perform experiments that require dynamic interactions with the dynamic system described by $A^{**}$, the EPM needs to build an Experimental Frame (EF), which is a model of the dynamic system itself, and which is simulated at the same time as the $A^{**}$ model. We explain in (Bonté, Duboz, Quesnel, and Müller 2009) how such EPM can be built by using a recursive simulation technique. Another specification which takes better benefice of the EF concept is described in (Bonté 2011). The EPM enables to specify this special interaction between the dynamic system described by the $A^{**}$ model and those described by the models of the triad sub-systems.

Finally, the $B^*$ model is reduced to the decision process that the observer $B$ performs to influence the SPOC, according to information obtained from the experiments realised with the $A_B$* model.

A proposal for a general structure of $T^*$ using DEVS is presented in figure 3. The rectangular boxes represent dynamic system models, specified either as atomic DEVS models or composed DEVS models. The lines between boxes represent connections between models that can be either "port $\rightarrow$ port" connections or "model $\rightarrow$ model" connections (meaning one or several "port $\rightarrow$ port" connections). We did not represent any connection within the $\rho_o$* model because the Observation and Action sub-models are just given as an example of the SPOC. The decision process model receives information from the $\rho_m$* model and can influence the $\rho_o$* model behaviour. The $\rho_m$* model is an experimentation process model. It embeds the $A^{**}$ model and experiments it using an experimental frame.

## 4 EXAMPLE OF APPLICATION OF THE FRAMEWORK

### 4.1 Introduction: Evaluation of model use in epidemiology

As emphasized previously, some systems cannot be experimented in the sense that we cannot set (or sometimes even observe) the input signal, and we may have very poor information about the monitoring system. Epidemics spreading in a human or animal population is a good example because modelling and simulation is widely used in epidemiology and ethical concerns prevent experimentation. In order to discuss the interest of our framework we explain how it can be used in the context of animal epidemiology.

Many of the processes involved in diseases spread are identified and are similar from an epidemics to another. However, the qualitative knowledge of theses processes is not sufficient to predict the system evolution. An accurate knowledge of the relative weights of each process is necessary. The problem is that this knowledge will be available only after the epidemics has occurred. Moreover, people may neither just let some disease spread with no reaction. Consequently the observer is part of the system and cannot stay passive. The surveillance system must be considered in any disease spread analysis (Höhle, Paul, and Held 2009) because the epidemics itself cannot be directly monitored. The issue is that the epidemics dynamics may depend on the surveillance system designed to observe it.

With the increasing use in M&S in epidemiology, we have many information on triads used in this field and some systematic reviews have been published (Singer 2010, Singer, Salman, and Thulke 2011). Considering the target system $A$, most of the processes involved in disease transmission and spread have been precisely described, leading to precise characterisation of epidemics systems considered at different scales. Considering the model$A_B$*, huge work has been done to simulate disease spread and several classes of models exist (Keeling and Rohani 2007). Considering the observer $B$, it is composed of the network of decisional institutions (such as OIE, national veterinary or human health departments) and research teams. We can observe how the decision process tells which management policy must be applied according to information gathered from the use of model $A_B$*. The $\rho_o$ relation between the observer and the target system is precisely described as a combination of a surveillance system and a control system. Considering the $\rho_m$ relation between the observer and the model, many kind of experimental plans have been designed

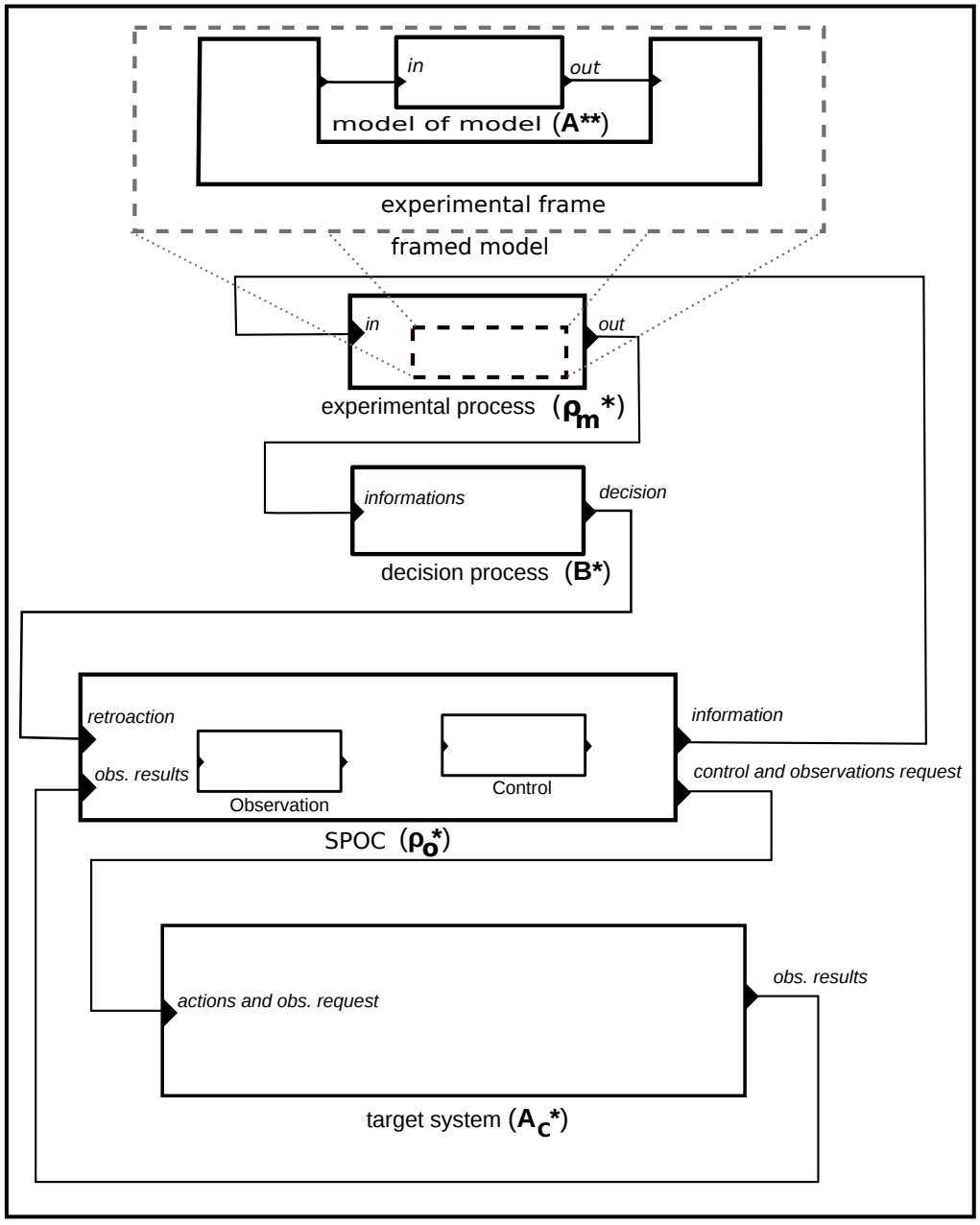


Figure 3: The T* DEVS structure.

and are described in the literature in order to perform calibration, optimisation or sensitivity analysis for instance.

As explained previously, the models themselves can hardly be evaluated in regards to their capacity to reproduce the target system behaviour. Nevertheless, we can evaluate the use of specific models in specific situations. Thanks to all information concerning model use in epidemiology, we are able to build reliable simulation models of these situations. In this section, we present how the formal model presented in section 3 can be used to evaluate the model use in the case of animal epidemiology. We use the conceptual framework presented in section 2 to organize our discourse. Likewise, in the general case of triad of triad presented in figure 2 we consider three triads. The first triad is the $T_{uc}$ system (for Triad Under Control). It is an imaginary situation where the $A$ entity is an epidemic, the $B$ entity is a surveillance and control system and the $A^*$ entity is an epidemiological model. The second triad is the $T_{uc}$* model which is a model of the $T_{uc}$ system built in order to evaluate model use. Finally, the third triad is the $T_{uc}$' triad which is composed of the $T_{uc}$ system, the $T_{uc}$* model and the research questions we have on $T_{uc}$.

### 4.2 $T$: the $T_{uc}$ system

The $T_{uc}$ system is composed of a system $A$, a SPOC ($\rho_o$), a decision unit $B$ and a model experimentation process ($\rho_m$) using a model $A^*$. We will refer to the system $A$ as the epidemiological system. It is a set of a hundred epidemiological units (sub-regions of a geographical area of interest) connected to one another by an infectious contact network through which an infectious epidemiological unit can infect its acquaintances. The SPOC is a system for epidemiological surveillance and control and is composed of a passive observation system, a proactive observation system and a control system. The passive observation system observes all components of the epidemiological system until an outbreak is detected. When an outbreak is detected, the proactive observation system and control system are activated. The control consists in reducing the move of animals in the area of interest. Several levels of move restriction are defined corresponding to different intensity of control. At first a default level is chosen, then the control system can modify the level of move restriction following the decision of the decision system. Note that the level of control is known (it can be quantified as the number of animals allowed to be moved from a place to another for instance) but the impact it has on the disease spread is unknown. The proactive observation system observes at regular time step a representative sample of epidemiological units in order to estimate at each time step the prevalence[1] in the area of interest. This information is then sent to the model experimentation process. The model experimentation process uses a SIS compartment model (Anderson and May 1979) and consists in calibrating the model using available data (prevalence estimation from the observation system). An interesting feature of the SIS model is that its dynamics is characterized by the $R_0$ indicator (Keeling and Rohani 2007). When $R_0 > 1$, a stable state is reached corresponding to a non null proportion of infectious individuals in the population equal to $1 - 1/R_0$. On the other hand, if $R_0 < 1$ the proportion of infectious individuals will tend to zero. The decision system receives the calibrated model from the experimentation process and computes the corresponding value of $R_0$ from this model. The decision system decides to increase or decrease the control level according to the value of $R_0$.

### 4.3 $C$: question we have on $T_{uc}$

The issues we want to address about $T_{uc}$ as the observer $C$ of the $T_{uc}$' triad are the following:

1. What is the impact of the epidemiological surveillance system on the production losses due to the disease?
2. What is the impact of the model experimentation process on the production losses due to the disease?
3. How these impacts depend on the epidemiological system of interest?

[1]The prevalence in a population is the proportion of infected individuals in this population

Type 1 questions deal with the design of the passive and proactive observation systems ($\rho_o$ relation). Type 2 questions deal with the design of the model experimentation process ($\rho_m$ relation). Finally, type 3 questions deal with the compatibility of the surveillance, control and model experimentation processes ($\rho_o$ and $\rho_m$) to the nature of the epidemiological system of interest ($A$). All the questions deal with the consequences that the design of the surveillance, control and modelling of the disease may have on the disease (possibly considering the nature of the disease for type 3 questions). The $T_{uc}$ system is described in such a way that we are able to identify indicators that we can use to characterize different design modality of the surveillance, control and model experimentation processes on the one hand, and different scenarios of diseases on the other hand. We refer to these indicators as"factors". We are also able to identify indicators to evaluate the consequences of the disease in terms of production losses and to evaluate quantitatively the surveillance and control effort. We will refer to these indicators as "criteria" because they enable us to evaluate different surveillance system, control system and model experimentation processes.

In our case, we choose the transmission rate between epidemiological units as a factor that would characterize the epidemiological system. It can be measured as the mean spreading speed between two neighbour units. We choose the sampling time period of the proactive observation system as a factor that would characterize the design of the surveillance system, It is measured as the time step between two successive sampling. As a factor that would characterize the model experimentation process we choose the binary answer to the question: "do we know the value of the $\gamma$ parameter of the SIS model for this disease?" (see equations 1 and 2). We choose two criteria to characterize the production loss due to the disease. The first is the cumulative time of infected epidemiological units measured as the total number of infected epidemiological units multiplied by the time they have been infected. The second is the quarantine length of the whole geographical area measured as the time period between the first detected outbreak and the last detected outbreak. Finally, as a criteria that would characterize surveillance and control cost, we choose to measure the surveillance effort as the total number of epidemiological units sampled by the proactive surveillance system and the control effort as the integral of the control intensity.

### 4.4 $T^*$: the $T_{uc}$* model

The interest in building the $T_{uc}$* simulation model is double. First, some of the factors or criteria are not directly measurable in $T_{uc}$. It is the case for the cumulative time of infected epidemiological units for instance. Second, the $T_{uc}$* model enables us to realize proper experimental plans allowing to empirically evaluate the influence of the factor values over the criteria values. Due to lack of space and because our objective is less to present quantitative results than a methodology, we do not present all details of the $T_{uc}$* model. Note that these details are given, as well as a complete DEVS specification, in the PhD dissertation of the first author (Bonté 2011). However we must note some important points in order to discuss the simulation results. The structure of the $T_{uc}$* model is similar to the one presented in figure 3.

Concerning the model of the epidemiological system, note that each epidemiological unit is modelled as a DEVS model and that the infectious contact network is the connection graph between IN/OUT ports of these models. Each model of an epidemiological unit is a two states automaton whose states are either Susceptible (S) or Infectious (I). In state I, an epidemiological unit can infect its neighbours in state S with an infection rate noted $r_{inf}$. The passive observation process model is connected to all epidemiological units model and detects a switch to an I state with a given detection probability. The proactive observation process model connects itself at regular time step to a sample of epidemiological unit models. The number of sampled epidemiological units is computed at each observation time step according to the prevalence observed at the previous observation time step, a desired relative precision and a statistical formula ordinarily used to compute the sample size in epidemiological surveys. The model of control modifies the infection rate $r_{inf}$ of all epidemiological units (susceptible units may become infectious) by multiplying it by a factor chosen in a collection of numbers corresponding to different control levels. The $A^{**}$ model is the SIS model given by the equations 1, and 2. The experimental frame used consists in setting the initial state and parameters and to observe the dynamics of the I state variable.

$$\frac{dS}{dt} = \gamma I - \beta IS \quad (1)$$

$$\frac{dI}{dt} = \beta IS - \gamma I \quad (2)$$

where

$\{S,I\} \in \mathbb{R}^2$ are the two state variables (representing respectively the proportion of susceptible and infectious individuals in the population),
$\beta$ and $\gamma$ are two parameters (respectively the infection rate and the recovery rate).

The EPM implements a swarm particle optimisation algorithm which enables to estimate the values of $\beta$ (and possibly the value of $\gamma$ if the recovery rate is unknown) that enable the best fit between SIS simulation results and the time series observed by the proactive observation model. The decision model $B^*$, uses $\beta$ and $\gamma$ values to compute the $R_0$ indicator ($R_0 = \beta/\gamma$). If $R_0 > 1$, the control intensity is increased to the superior level. If $R_{0\_tol} < R_0 \leq 1$, the control is unchanged. If $R_0 \leq R_{0\_tol}$, the control intensity is decreased to the inferior level. We consider that the level of control is known by the decision maker but the corresponding factor applied to the infection rate $r_{inf}$ is unknown.

### 4.5 $\rho_{m\_c}$: simulation of $T_{uc}$*

The $T_{uc}$* model is stochastic so we can get several different simulation results using the same set of parameters and changing the random number generator seed of the simulator. In this section we comment the results of a single simulation. Figure 4 presents the simulation outputs for this simulation.

#### 4.5.1 Trajectory of the epidemiological system

On the a) chart, we see the evolution of prevalence in the model of epidemiological system (plain curve). The dashed curve shows an example of prevalence evolution for the simulation of the model of epidemiological system with no control. We observe that the epidemics ends when the system is controlled (prevalence is null at the end of the simulation for the plain curve), although in the case with no control, the disease become endemic (prevalence seams to become stable around a positive value of the dashed curve).

#### 4.5.2 Trajectory perceived by the surveillance system

Chart b) shows prevalence values estimated by the proactive observation model (dots). It corresponds to the prevalence observed in the samples. The "real" prevalence in the model of epidemiological system is plotted as a plain curve. We observe that both curves are very close. Distance in time between the estimated prevalence dots corresponds to the sampling time period of the proactive observation model. The date of the first prevalence estimation corresponds to the activation of the proactive observation model triggered by the first outbreak detection by the passive observation model. We notice that this activation occurs a short time after the epidemic starts (the plain curve is already increasing).

#### 4.5.3 Trajectory of an indicator of the proactive observation model activity

Chart c) shows the proportion of epidemiological units sampled at each sampling realised by the proactive observation model (recall that the number of units to sample is computed by the proactive observation model at each observation time step). We notice that variations are wide.

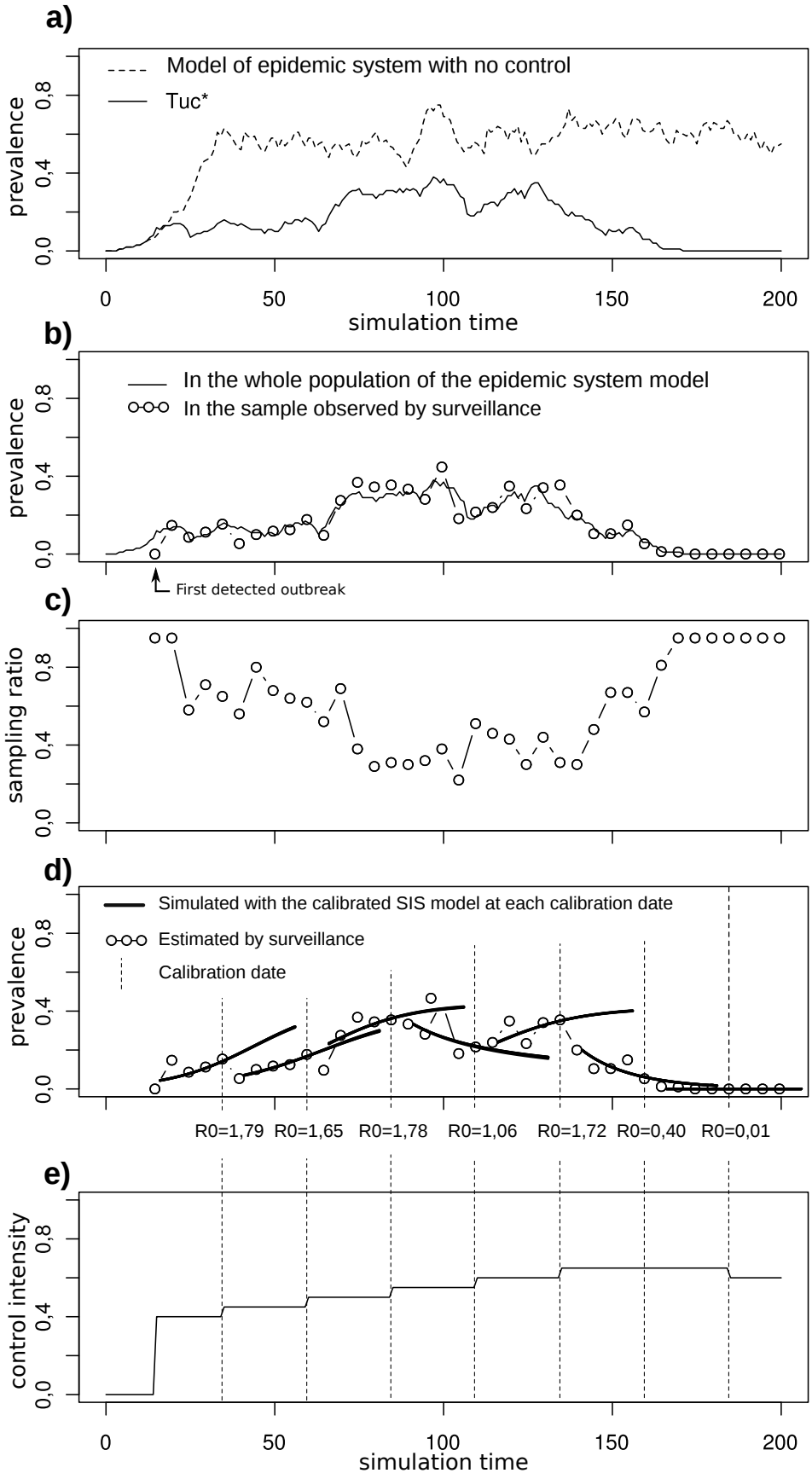


Figure 4: Simulation outputs of $T_{uc}$* model

### 4.5.4 Information brought by the EPM

Chart d) shows the results obtained by the EPM. The prevalence data estimated by the proactive observation model have been replotted (dots). Recall that calibrations performed by the experimentation process model are based on these data. The vertical dashed lines mark the dates at which calibrations occurs (the EPM perform a calibration each time 5 new prevalence data are available). For each calibration, the prevalence evolution simulated with the calibrated SIS model has been plotted (plain short curves) from the date of the first observation used for this calibration (five observation before), until a prediction horizon fixed to the time between two calibrations. The $R_0$ value computed for each calibration is written below each calibration date. Note that the prevalence simulated with the SIS model fits very well the surveillance data (the five prevalence observations preceding a calibration are almost superimposed with the corresponding SIS simulation curve), and predicts badly the future prevalence (observations following a calibration can be very far from the SIS simulation curve corresponding to this calibration). This last result is expected because the control level following a calibration may be different of the control level preceding the calibration.

### 4.5.5 Trajectory of an indicator of the control model activity

Chart e) shows the evolution of the control intensity during the simulation. Note that the control model is activated at the same time as the proactive observation model (the control intensity is 0 before the first prevalence value is observed on charts b), c) and d). At each calibration, the control level is revised. It is increased if the estimated $R_0$ is superior to 1 (which is true until the penultimate calibration), maintained if $R_{o\_tol} < R_0 < 1$ (which is true at the penultimate calibration), and decreased if $R_0 \leq R_{0\_tol}$ which is true for the last calibration.

### 4.5.6 Experimental plan on $T_{uc}$*

In a M&S approach, we transfer the questions we have on $T_{uc}$ to $T_{uc}$* and we perform an experimental plan on $T_{uc}$*. We performed a light experimental plan on $T_{uc}$* in order to show that we can formulate questions of type 1, 2 and 3 presented in section 4.3 as experiments on $T_{uc}$*. This is an empirical approach leading to statistically measure the influence of our factors on our criteria.

Notice that we can compute our criteria from simulation outputs. For instance, the quarantine duration criterion can be measured on the chart a) of figure 4 and the surveillance effort criterion can be measured on chart c). For a given set of parameters and a few modalities tested on our factors, we performed thirty simulations for each factors combination. As an answer to "type 2" and "type 3" questions, we could show that for the tested modalities, knowing the value of the $\gamma$ parameter (fixing $\gamma$ and calibrating only $\beta$ instead of calibrating both $\beta$ and $\gamma$ parameters with the EPM) has no significant impact on the cumulative infected time in the scenario of a slow disease spread but had a significant impact in the case of a fast disease spread.

For the same set of parameters we also found that decreasing the proactive sampling time period significantly decreased our surveillance effort indicator for a same value of our control effort indicator. This result is counter intuitive because more epidemiological units are sampled by time units if sampling time period is lower. This is due to the fact that epidemics are in average shorter if the sampling is more frequent (control is more efficient). This kind of answers to questions of type 1 give a different (an we think interesting) point of view on monitoring systems which are usually evaluated on their capacity to capture the epidemic trend and not as a part of the epidemics dynamics.

## 4.6 $\rho_{o\_c}$: potential answers on $T_{uc}$

### 4.6.1 Validation of $T_{uc}$*

Our motivation to build the model $T_{uc}$* is that the model $A_B$* cannot be validated. However, note that the system $T_{uc}$ contains the target system $A$. Consequently, we could think we would not be able to build a

validated $A_C$* model. At that point, it is important to notice that the questions we want to address with the $A_B$* model are not the same that those we want to address with the $A_C$* model. In the $T_{uc}$ triad, the $A_B$* model is used to produce a summary of the $A$ system (the $R_0$ indicator in our case). In the $T_{uc}$' triad, the $A_C$* model is used to reproduce the complexity of the $A$ system in order to evaluate if the SIS model is able to produce a satisfactory summary of the disease dynamics. The summary is considered satisfactory depending on the efficiency of the control that it enables to perform. In the case of the $T_{uc}$* model we presented, we consider that the $A_C$* is valid to represent the complexity due to non-homogeneous mixing of the population. Indeed, epidemiological units are connected via a network of infectious contact that may not be random (we choose a 2D regular lattice network for the shown simulations). Consequently, $T_{uc}$* is valid if we use the $A_C$* model as a sufficient informative hypothesis. Then, under the hypothesis of $A_C$*, the answers we have by experimenting $T_{uc}$* can be transferred to $T_{uc}$. For this reason, we think that the most interesting questions to address with the $T_{uc}$* model are those of type 3, i.e. evaluating different types of models integrated in different situation of disease management.

### 4.6.2 Learning on $T_{uc}$ and offered perspectives

The example of application given in this paper only showed that, under the $A_C$* hypothesis, model evaluation can be done depending on the efficiency of the control the model allows. Intensive experimental plans must now be performed to bring reliable results to the epidemiological modelling community. The first type of results would be recommendations about which kind of model (spatial or not, aggregated or individual based) and model experimentation process could be used depending on the epidemics situation (speed or low infection rate of the disease, availability of surveillance effort, ... ). The second type of results is to help designing new $T_{uc}$ systems that cannot have been designed already because they cannot have been tested yet. We think that experimenting on $T_{uc}$* can help to design new surveillance and control systems based on simulation model results. Finally, $T_{uc}$* can support epidemiologists formation by reproducing some of the mechanisms leading to wrong model predictions.

## 5 DISCUSSION

We think that the contribution of this work can be considered as two distinct conceptual tools. The first contribution is a methodology based on the conceptual framework presented in section 2, illustrated figure **??** and instantiated section 4. The base of this methodology is to use the TMS to enable a reflexive study of TMS activity in order to improve it in its most problematic cases. We hope that this methodology will be used for many other applications and developed further. The second contribution is the model $T^*$ presented in section 3. This paper only draws the main lines of a generic $T^*$ model (see figure 3) but we think that it offers a sufficient framework to many improvements based on all work previously done in TMS, notably considering all work done on experimental frame specification and uses. We think the EPM presented in (Bonté, Penot, Page, and Tourrand ress) is generic but it undoubtedly needs to face more applications. Note that using DEVS formalism to specify $T^*$ allows to build triads using existing models, which is particularly interesting for $A_C$* and $A$**.

Finally a third contribution is the software tool to build the $T^*$ simulator. All computer developments used for simulations have been done using the Virtual Laboratory Environment (Quesnel, Duboz, and Ramat 2009). A package called "experimenter" has been developed for the EPM but further development remain to be done before publishing the generic "t-star" package.

## 6 CONCLUSION

We proposed a conceptual and formal framework to evaluate models of systems that cannot be experimented. For these systems, a validation process based on comparing system and model behaviours within an experimental frame is meaningless. Thus model evaluation must be done on other criteria. We proposed to model the whole Minsky triad composed of three entities: the object (or target system), the observer

and the model. Doing so, we represent the feedback loop between decision made using a model of a target system, and the target system itself. Such a framework can be used to model, and then to test, *a priori* decision making process in a context where experimentation is not possible.

The Minsky triad is here implemented as a dynamic system. Therefore, we can use the general framework proposed by the TMS. We can then experiment the triad and evaluate not only the model but the situations involving the three interacting entities of the triad. This may be the only way to address some essential questions about the use of models for decision making in crisis management. We emphasized this issue with the example of model used for epidemics management and we are convinced that this work shall give rise to a new kind of model evaluation in contexts where experiment is not possible, such as financial crisis management, epidemiology, or climate change for instance. We hope that new kinds of model based decision support will arise thanks to a better evaluation of these models and models uses.

## REFERENCES


Anderson, R. M., and R. M. May. 1979. "Population biology of infectious diseases: Part I and II". *Nature* 280:361–367.

Bonté, B. 2011, december. *Modélisation et simulation de l'interdépendance entre l'objet, l'observateur et le modèle de l'objet dans la Triade de Minsky. Application à la surveillance épidémiologique en santé animale. (Modelling and simulation of interdependence between the object, the observer and the model of the object in the triad of Minsky. Application to animal health surveillance).* Ph. D. thesis, Université Montpellier II. PhD in french with summary in english.

Bonté, B., R. Duboz, G. Quesnel, and J.-P. Müller. 2009, July 13th-16th. "Recursive simulation and experimental frame for multiscale simulation". In *SCSC'09: Summer Computer Simulation Conference*.

Bonté, B., É. Penot, C. L. Page, and J.-F. Tourrand. 2011 (under press). *modélisation des exploitations agricoles avec Olympe (Seconde édition)*, Chapter Liaison d'un outil de modélisation d'exploitations agricoles (Olympe) avec une plateforme de modélisation multi-agents (Cormas). Quae.

Höhle, M., M. Paul, and L. Held. 2009. "Statistical approaches to the monitoring and surveillance of infectious diseases for veterinary public health". *Preventive Veterinary Medicine* In Press, Corrected Proof:–.

Keeling, M., and P. Rohani. 2007. *Modelling Infectious Diseases In Humans And Animals*. Princeton University Press.

Minsky, M. 1965. "Matter, Mind and Models". In *Proceedings of IFIP Congress*, 45–49.

Quesnel, G., R. Duboz, and É. Ramat. 2009, April. "The Virtual Laboratory Environment – An operational framework for multi-modelling, simulation and analysis of complex dynamical systems". *Simulation Modelling Practice and Theory* 17:641–653.

Alexander Singer 2010. "External report reviewing the previous opinions of the Panel on Animal Health and Welfare concerning the application of quantitative tools, in the sequence of the current self mandate on "Good Practice in Conducting Scientific Assessments in Animal Health Using Modelling"". SCIENTIFIC REPORT submitted to EFSA (Question No EFSA-Q-2009-408).

Singer, A., M. Salman, and H.-H. Thulke. 2011. "Reviewing model application to support animal health decision making". *Preventive Veterinary Medicine* In Press, Corrected Proof:–.

Traore K. Mamadou, M. A. 2006. "Capturing the dual relationship between simulation models and their context". *Simulation Modelling Practice and Theory* 14:126–142.

Zeigler, B. P., T. G. Kim, and H. Praehofer. 2000. *Theory of modeling and simulation: Integrating Discrete Event and Continuous Complex Dynamic Systems*. Academic Press.